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Journal of Acute Medicine 2 (2012) 117–120

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Case Report

Onion-induced anaphylactic shock rapidly evolving to allergic right ventricular myocardial infarction and subsequent cardiogenic shock

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Received 31 March 2012; accepted 10 August 2012
Available online 15 October 2012

Abstract

The type II variant of Kounis syndrome is defined as a rare allergic myocardial angina or infarction event in patients with preexisting quiescent coronary artery disease. Various causative factors have been implicated in the etiology of Kounis syndrome. However, reports highlighting the importance of recognizing a decreased preload caused by allergic right ventricular (RV) myocardial infarction and subsequent cardiogenic shock from ongoing anaphylactic shock are rare. Here we report the case of a 54-year-old male who initially presented with anaphylactic shock after ingesting onions. His condition silently progressed to RV infarction and cardiogenic shock within 2 hours of symptom onset. Under such instances, it is crucial to promptly identify RV infarction and cardiogenic shock by repeatedly performing electrocardiography at frequent intervals.

Keywords: Allergic angina; Allergic myocardial infarction; Kounis syndrome; Anaphylactic shock; Cardiogenic shock; Onion

1. Introduction

The type II variant of Kounis syndrome is defined as a rare, irreversible, allergic myocardial angina or myocardial infarction (MI) event in patients with preexisting quiescent coronary artery disease (CAD). In cases where the allergic myocardial injury involves the right coronary artery, subsequent occurrence of sudden cardiogenic shock is possible. Management of anaphylactic shock differs from that of MI—the former requires massive fluid resuscitation along with anti-allergen and vasopressor therapy, whereas the latter when accompanied with subsequent cardiogenic shock requires emergency percutaneous coronary intervention and intra-aortic balloon pump (IABP) implantation to maintain hemodynamic stability. Determining the cause of hemodynamic instability in patients with type II Kounis syndrome is challenging for even the most seasoned clinician, and inaccurate or delayed management may lead to fatal outcomes.

2. Case report

A 54-year-old man of medium build (170 cm in height, 88 kg in weight) presented at the emergency department of our institution with dizziness, weakness, chest tightness, and generalized pruritus with skin erythema. All these symptoms occurred in approximately 20 minutes after he ate onions at dinner. His medical history revealed hypertension, CAD, and anterior MI, for which he had undergone coronary artery bypass graft surgery 2 years earlier. In addition, he was known to be allergic to onions, green onions, and garlic. However, the previous allergic events had been mild and resolved soon after the intake of oral fexofenadine hydrochloride (Allegra).

On initial examination, his initial vital signs were as follows: body temperature, 37°C; heart rate, 55 beats per minute (bpm); respiratory rate, 20 bpm; and blood pressure,

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2211-5587/$ - see front matter
http://dx.doi.org/10.1016/j.jacme.2012.08.002

50/31 mmHg. On physical examination, he seemed irritable and listless but alert. Urticaria and increased temperature was observed all over his face, trunk, and extremities. Chest auscultation did not indicate stridor, wheezing, or basal crackles. Based on a presumed diagnosis of anaphylactic shock, oxygen was supplied via a mask at 8 L/min; in addition, fluid resuscitation, intravenous diphenhydramine, methylprednisolone, and ranitidine were immediately administered. Epinephrine was withheld considering the patient's cardiovascular history and the associated risk of adverse events, namely, epinephrine-induced vasospasm or MI. Initial electrocardiography (ECG) revealed first-degree atrioventricular block and right bundle branch block that was consistent with an ECG finding obtained 3 years earlier (Fig. 1).

Chest X-ray revealed mild symmetric infiltrates with borderline cardiomegaly. Laboratory data indicated leukocytosis (16,800/μL) with a normal differential count and an eosinophil count of 0.4%. The glutamate oxaloacetate transaminase level was 26 U/L (5−35 U/L). Initial cardiac enzyme levels measured within 70 minutes after onion exposure were within the normal range as follows: creatine kinase (CK), 134 U/L (39−308 U/L); CK-MB, 21 U/L (7−25 U/L); and troponin I, 0.086 μg/L (0−0.5 μg/L).

The patient remained hypotensive (89/51 mmHg) even after 2.5 L of normal saline fluid administration. Central venous catheterization was then performed to maintain hemodynamic stability, and the initial pressure was high (28 cm H_2O). Although the generalized skin rash disappeared, the patient continued to complain of progressive chest compression without radiation to any body parts. ECG obtained 2 hours after the allergic event revealed ST-segment elevation in leads II, III, and aVF (Fig. 2). Cardiac enzyme levels at this point were as follows: CK, 550 U/L; CKMB, 104 U/L; troponin-I, 1.926 μg/L. There was no significant ST-segment elevation in lead V4R on a right-sided ECG. Acute ST-segment elevation with inferior wall MI was diagnosed, and primary cardiac intervention was performed immediately. Considering that anaphylaxis may not have been the only component of shock, continuous infusions of norepinephrine and dopamine were administered for the management of strongly suspected cardiogenic shock. With this treatment, the patient's blood pressure was maintained at 119/69 mmHg, with a heart rate of 104 bpm. Aspirin (300 mg) and clopidogrel (400 mg) were administered in combination with continuous heparin infusion before primary catheterization. Morphine was not used to avoid mast cell activation and additional histamine release.

Selective cardiac angiography revealed total occlusion of the distal right coronary artery. Angioplasty using a voyager balloon was successfully performed (Fig. 3). The saphenous vein graft was patent to the diagonal branch and the obtuse marginal branch, whereas the left internal mammary artery graft was patent to the left anterior descending artery. An IABP was implanted and set at a rate of 1:1. A temporary pacemaker was also inserted and set at 60 bpm for the management of cardiogenic shock. In addition, intravenous antihistamine and hydrocortisone were regularly administered during admission.

CK (7257 U/L) and CK-MB (551 U/L) levels peaked approximately 7 hours after catheterization, and ECG revealed a Q wave in the inferior lead (Fig. 4). Two days after the primary cardiac intervention, the IABP and temporary pacemaker were removed under continuous infusion of dobutamine. ECG performed 2 days later revealed a marked decrease in left ventricular ejection fraction (39%) and poor left ventricular contractility, with posterior and apical wall hypokinesis. The patient was transferred to the general ward on the 3rd hospital day because of a relatively stable hemodynamic state. However, he developed fever, dyspnea, and productive cough the following day, and nosocomial pneumonia was diagnosed. Three days after piperacillin/tazobactam treatment, i.e., on the 7th hospital day, the patient took discharge against the physicians' advice with a prescription of oral antibiotic therapy.

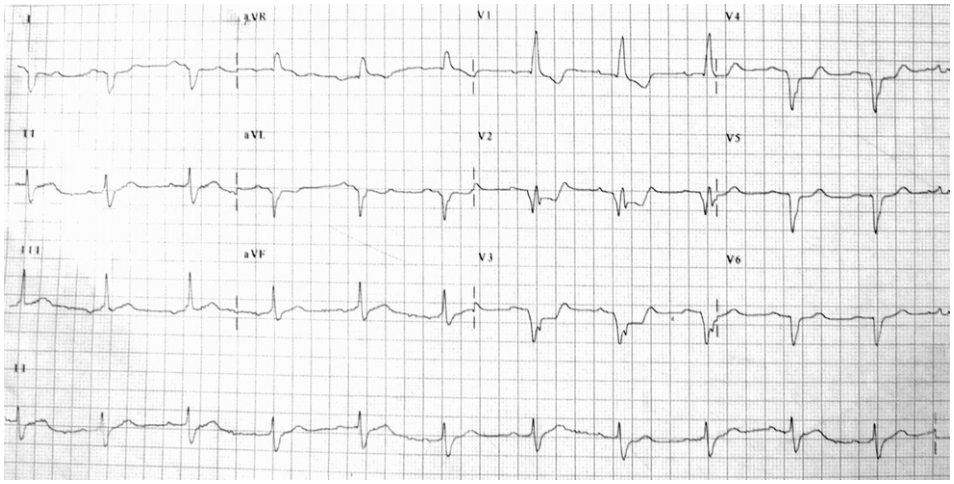

Fig. 1. Initial electrocardiography (ECG) showed no interval change compared to the last ECG three years ago.

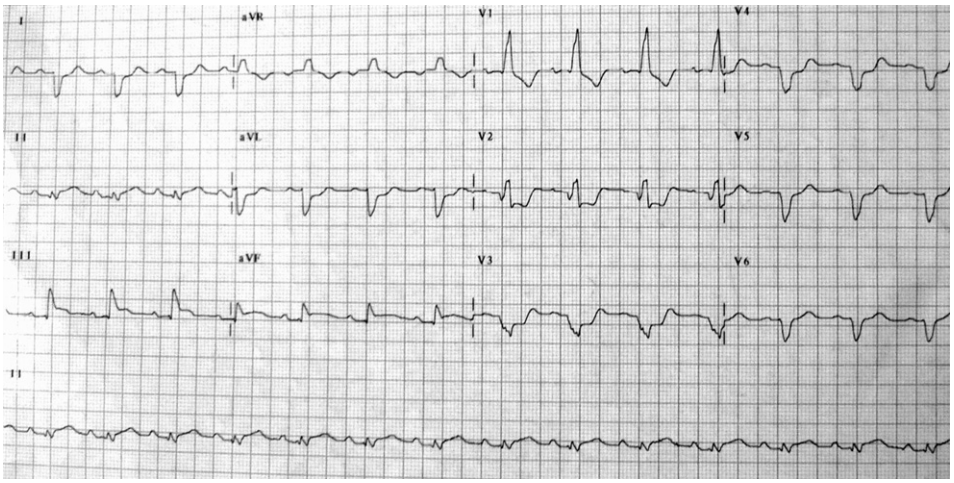

Fig. 2. Two and a half hours later, repeated ECG exhibited II, III, aVF ST-segment elevation myocardial infarction.

3. Discussion

Diverse allergic agents responsible for Kounis syndrome have been reported in the literature, including nonsteroidal anti-inflammatory drugs, antibiotics, proton pump inhibitors, muscle relaxants, chemotherapeutic agents, drug-eluting stents, insect stings, shellfish ingestion, tetanus immunization, and venoms.[1–7] Although allergic angina proves to be only vasospasms in a majority of cases, and primary catheterizations show no significant stenosis, type II Kounis syndrome involves the occurrence of true MIs that are capable of presenting as cardiogenic shock and lethal dysrhythmias. For a patient with a preexisting history of quiescent CAD, a tiny atherosclerotic plaque rupture may cause a significant MI event.[7] Such an event may occur any minute after an anaphylactic reaction has initiated.

Morphine sulfate is prescribed as adjunctive therapy in patients with acute coronary syndrome; however, it may cause histamine release by activating mast cells and may not be suitable for the management of allergic MI.[8,9] Epinephrine, on the other hand, is considered as an essential treatment to reverse anaphylactic effects. However, serious adverse effects, including coronary spasm and MI, are also reported to be directly related to epinephrine.[10–13] Epinephrine is a potent vasoconstrictor that can maintain systemic vascular resistance during anaphylactic shock; however, this property may also induce coronary vasospasm and myocardial injury. Although epinephrine is considered as the first-line treatment for anaphylactic shock, its benefit in patients with allergic MI is controversial.

Chest tightness is considered a symptom of an allergic reaction.[14] ECG may be performed routinely in patients who present with chest discomfort, unstable hemodynamics, or preexisting cardiovascular disease. In our case, after desensitizing management, persistent chest pain and hypotension raised a suspicion of acute coronary syndrome. A single ECG was apparently not sufficient to detect this evolution; therefore, repeated ECG performance within short intervals of time was essential. On the basis of the rapid evolution of allergic angina to true ST-segment MI in this case, we suggested following the Joint Classification Committee/American Heart Association guidelines for patients presenting with allergic reactions and persistent chest discomfort, particularly for those with a preexisting history of cardiovascular disease.[15]

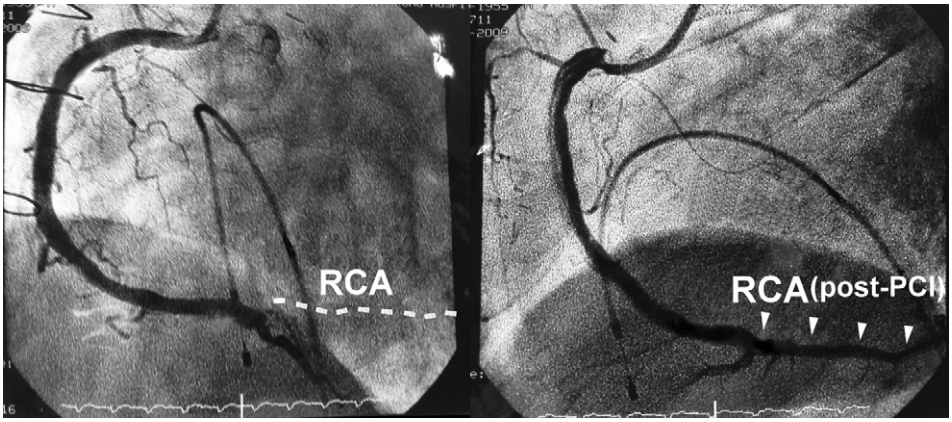

Fig. 3. Before: selective cardiac angiography revealed RCA total occlusion; after voyager balloon angioplasty, RCA gained perfusion successfully.

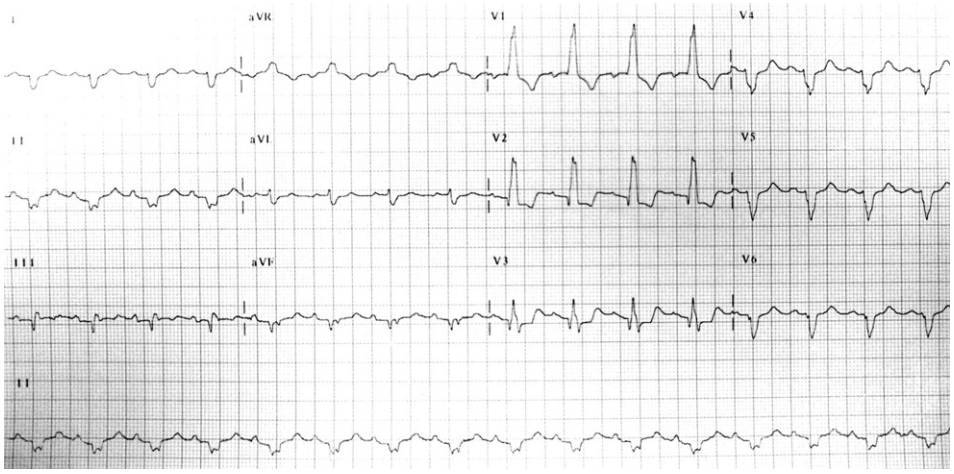

Fig. 4. Post-PCI ECG showed Q wave in inferior lead.

As per our knowledge, this case is unique because the allergic MI evolved rapidly after exposure to the allergen; furthermore, initial ECG and cardiac marker analysis at presentation were not definitive. The risk of new-onset allergic MI should be highlighted in patients with significant cardiovascular compromise even if initial screening tests are negative. Lethal cardiogenic shock or dysrhythmias may be overlooked if anaphylactic shock is considered the sole basis for treatment.

4. Conclusion

In conclusion, when a patient with anaphylactic shock presents to the emergency department with profound shock or persistent chest pain despite desensitizing management, repeating ECG and cardiac marker analysis at short intervals of time are important to ensure that acute coronary syndrome does not occur simultaneously.

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